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Original Research Article

Exploring the relationship between triglyceride-glucose index and peripheral neuropathy in Nigerian patients with type 2 diabetes: a comparison with other metabolic markers

Blessing Kenechi Myke-Mbata[1], Bruno Basil[2]*, Izuchukwu Nnachi Mba[3], Terna Gav Ambrose[1]

[1]Department of Chemical Pathology, Rev. Fr. Moses Orshio Adasu University, Makurdi, Nigeria
[2]International Institute for Pathology and Forensic Science Research, David Umahi Federal University of Health Sciences, Uburu, Nigeria
[3]Department of Chemical Pathology, Nile University, Abuja, Nigeria

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***Correspondence:**
Dr. Bruno Basil,
E-mail: basilb@dufuhs.edu.ng

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ABSTRACT

Background: Diabetic peripheral neuropathy (DPN) is a common and disabling complication of type 2 diabetes mellitus (T2DM). The triglyceride–glucose (TyG) index, a surrogate marker of insulin resistance, has been associated with several diabetes-related complications, but its role in identifying DPN remains unclear. This study evaluated the relationship between the TyG index and DPN among Nigerian patients with T2DM and compared its performance with other metabolic markers.
Methods: This hospital-based cross-sectional study was conducted among adults with T2DM attending diabetes clinics in two tertiary hospitals in North-Central Nigeria. DPN was assessed using the Michigan Neuropathy Screening Instrument, 10-g monofilament and 128-Hz tuning fork. The TyG index, albumin–creatinine ratio (ACR), glycated haemoglobin (HbA1c), serum uric acid and apolipoprotein A-I (Apo A-I) were measured.
Results: The prevalence of DPN was 61.8% (n=63). DPN was associated with longer diabetes duration, higher hypertension prevalence, lower physical activity and worse renal indices. Participants with DPN had lower Apo A-I ($p=0.013$), higher urine albumin ($p<0.001$) and higher ACR ($p<0.001$). The TyG index did not differ significantly between groups ($p=0.218$) and showed poor diagnostic performance (AUC 0.427, $p=0.218$). By contrast, ACR (AUC 0.757, $p<0.001$) and Apo A-I (AUC 0.647, $p=0.013$) demonstrated better discriminatory ability.
Conclusions: TyG index showed poor diagnostic utility for DPN, whereas ACR and Apo A-I were significantly associated with neuropathy and provided superior discrimination. These findings support the use of renal and lipid-related markers alongside routine neuropathy screening.

Keywords: Albumin-to-creatinine ratio, Apolipoprotein A-I, Diabetic peripheral neuropathy, Nigeria, Type 2 diabetes mellitus, Triglyceride–glucose index

INTRODUCTION

Type 2 diabetes mellitus (T2DM) represents one of the most pressing public health problems worldwide and accounts for more than 90% of all cases of diabetes.[1] According to estimates from the International Diabetes Federation, 537 million adults were living with diabetes in 2021 and this number is expected to rise to 783 million by 2045.[2] Much of this increase is occurring in low- and middle-income countries. Across sub-Saharan Africa, rapid urbanisation, population ageing and shifts toward sedentary lifestyles and calorie-dense diets have driven a steep rise in T2DM prevalence.[3] Nigeria, as the region's most populous nation, carries a substantial share of this

burden, with large numbers of affected individuals who are either undiagnosed or sub-optimally treated. Many patients therefore present to hospital with advanced disease and established organ damage, while tertiary facilities struggle with overcrowding, prolonged waiting times and limited opportunities for sustained clinician–patient engagement.[4] These realities underline the importance of tools that can support earlier identification of high-risk patients within routine clinical practice.

The clinical impact of T2DM is largely determined by its long-term complications. These include microvascular conditions such as retinopathy, nephropathy and neuropathy, as well as macrovascular diseases including ischaemic heart disease, stroke and peripheral arterial disease.[5] Among these, DPN is particularly disabling, contributing to chronic pain, sensory impairment, gait disturbance and a heightened risk of foot ulceration and lower-limb amputation.[6] A range of interrelated biological processes, including sustained hyperglycaemia, insulin resistance, oxidative stress, inflammation and endothelial dysfunction, promote progressive neural and microvascular injury.[7] Identifying individuals at increased risk of neuropathy before irreversible nerve damage has occurred is therefore a central goal of diabetes care.

Insulin resistance is a key driver of metabolic dysregulation in T2DM and plays an important role in the development of its complications, including peripheral neuropathy. However, direct measurement of insulin resistance is technically demanding and rarely feasible in routine clinical settings.[8] This has led to the development of surrogate indices derived from standard laboratory tests. The TyG index, calculated from fasting triglyceride and fasting glucose concentrations, has been widely validated as a practical proxy for insulin resistance and has been linked to cardiometabolic risk in multiple populations.[9] These properties have generated interest in its potential utility for identifying patients at risk of diabetic complications, including DPN. Its simplicity and low cost make it especially appealing for use in resource-constrained settings, although available evidence remains inconsistent and data from African populations are scarce.

Beyond the TyG index, several routinely measured biochemical markers provide insight into the mechanisms underlying diabetic complications. The albumin–creatinine ratio (ACR) reflects early renal injury and systemic endothelial dysfunction and is strongly associated with both nephropathy and cardiovascular disease.[10] Glycated HbA1c represents long-term glycaemic exposure and is a well-established predictor of microvascular and macrovascular outcomes.[11] Elevated serum uric acid has been linked to oxidative stress and vascular dysfunction, processes that contribute to progressive metabolic and microvascular damage.[12] Apolipoprotein A-1 (Apo A-1), the principal protein component of high-density lipoprotein cholesterol, is a key mediator of reverse cholesterol transport and vascular protection and is inversely related to cardiovascular risk.[13] Because these markers reflect distinct but overlapping pathogenic pathways, their comparison with the TyG index provides a useful framework for assessing the latter's clinical relevance in neuropathy risk stratification.

Although studies from other regions support a biological link between the TyG index and vascular or metabolic risk, its value for predicting microvascular complications such as DPN in African populations remains uncertain.[14,15] This knowledge gap is particularly important in Nigeria, where delayed presentation and limited resources contribute to poor outcomes. A low-cost marker capable of identifying patients at increased neuropathy risk could therefore have significant clinical impact. The present study was designed to evaluate the utility of the TyG index as a marker of peripheral neuropathy among Nigerian adults with T2DM and to compare its diagnostic performance with that of other commonly used metabolic indicators.

METHODS

Study design and setting

This was a hospital-based, cross-sectional study conducted at the endocrinology and diabetes outpatient clinics of two major tertiary health institutions in North-Central Nigeria. The cross-sectional design allowed for simultaneous assessment TyG index and DPN in the study population, providing preliminary but clinically relevant insights to guide larger longitudinal studies. The study was conducted at the Benue State University Teaching Hospital, Makurdi and the Federal Medical Centre, Makurdi, Benue State, Nigeria. These hospitals are major referral centres serving both urban and rural populations, with a combined catchment area of approximately 2–3 million people. The study was carried out between February and September 2019, during which consecutive patients with T2DM attending follow-up visits were approached for recruitment.

Ethical considerations

Ethical approval for the study was obtained from the Benue State University Teaching Hospital Health Research Ethics Committee with reference number BSUTH/MKD/HREC/2013B/2017/0003. Written informed consent was obtained from all participants before enrolment, after a full explanation of the study objectives and procedures in English or the local language (Tiv and others). Participation was voluntary and participants were informed of their right to withdraw at any stage without loss of clinical care. The study was conducted in accordance with the Declaration of Helsinki (2013 revision).[16] To further maintain participants' confidentiality, hard-copy data were stored in locked cabinets while electronic data were password-protected, accessible only to the study team.

Sample size determination and participant recruitment

The required sample size for this study was derived using the reported prevalence of diabetic peripheral neuropathy among a population of Nigerian patients with T2DM. A previous hospital-based study documented a prevalence of 82%, which was adopted for sample size estimation.[17] Using this prevalence and applying a 95% confidence level with a 5% margin of error, the estimated minimum sample size was 227 participants. To account for possible non-response and incomplete data, an additional 10% was included, giving a final required sample size of approximately 250 participants. A total of 100 participants were recruited. This pragmatic sample size was considered adequate for exploratory analyses of diagnostic accuracy and hypothesis generation.[18]

Participants were eligible if they were adults (≥18 years) with a confirmed diagnosis of T2DM based on American Diabetes Association (ADA) criteria and had attended the diabetes clinic for at least 6 months.[19] Exclusion criteria included type 1 diabetes, gestational diabetes, severe intercurrent illness, decompensated cardiac failure, known chronic liver disease, active infection, pregnancy or refusal to give consent. Recruitment was by consecutive sampling of all eligible patients presenting during the study period until the required sample size was achieved. A recruitment log was maintained to track eligible, consenting and excluded participants, ensuring transparency and reproducibility.

Data collection and clinical measurements

Data were collected by trained research assistants using a research proforma. Information obtained included socio-demographic variables (age, sex, education, occupation, marital status), medical history (duration of diabetes, comorbidities, medication history, smoking, alcohol use, family history of diabetes) and lifestyle factors. Data collectors underwent a 2-day training to ensure uniformity and reduce inter-observer variability.

Anthropometric measurements were obtained following standardized protocols; height and weight were measured with participants wearing light clothing and no shoes, using a SECA weighing scale with height attachment. Body mass index (BMI) was calculated as weight (kg) divided by height squared (m^2). Clinical measurements including blood pressure were recorded using a calibrated mercury sphygmomanometer with two readings taken five minutes apart and averaged. If the two readings differed by more than 10 mmHg, a third measurement was taken and the mean of the two closest values was used. Hypertension was defined as Systolic BP ≥140 mmHg, diastolic BP ≥90 mmHg or current use of antihypertensive medications.[20]

Biochemical assays

Venous blood samples were collected after an overnight fast of 8–12 hours. Approximately 5 ml of venous blood was drawn from the antecubital vein or any other accessible vein under aseptic conditions. Samples were centrifuged at 5000 rpm for 5 minutes within 2 hours of collection and plasma/serum aliquots were stored at –20°C until analysis. Glucose, HbA1c, uric acid, creatinine and lipid parameters were measured using the Cobas c311® automated analyser (Roche Diagnostics), while APO A1 and Albumin were quantified using immunoturbidimetric assay kits (with intra- and inter-assay coefficients of variation <5%). To ensure accuracy, all assays were performed in duplicate and quality control sera were run daily in the laboratory. The laboratory participates in external quality assurance programmes.

The TyG index was calculated as the natural logarithm of the product of fasting triglyceride concentration and fasting glucose concentration (both in mg/dk), divided by 2, while ACR was calculated by dividing the urinary albumin concentration (mg/l) by the urinary creatinine concentration (g/l) and expressed as milligrams of albumin per gram of creatinine (mg/g).[21,22] Poor glycaemic control was defined as HbA1c ≥7.0% (53 mmol/mol).[23]

Assessment of peripheral neuropathy

Peripheral neuropathy was assessed using the validated Michigan Neuropathy Screening Instrument (MNSI), which comprises a 15-item self-administered questionnaire and a structured physical examination of the lower extremities.[24] The physical assessment included inspection for deformities, dry skin, calluses, infections, fissures and ulcers, along with evaluation of vibratory sensation and ankle reflexes. A questionnaire score of 7.0 or more and a physical assessment score of 2.0 or more were considered abnormal. In addition, 10g monofilament testing and 128-Hz tuning fork vibration testing were performed on the plantar surfaces of both feet to improve diagnostic accuracy. Patients with at least two abnormal test results (MNSI, monofilament, vibration or ankle reflex) were classified as having DPN. These findings were supplemented with documentation from the patients' clinical records, including any history of neuropathic symptoms such as numbness, tingling or burning sensations. Other diabetic complications such as nephropathy, retinopathy and macrovascular disease were not included as study outcomes but were documented as covariates where relevant.

Statistical analysis

Data were entered into a secure database and analysed using SPSS version 25 (IBM Corp., Armonk, NY, USA). Continuous variables were assessed for normality using the Shapiro–Wilk test. Normally distributed data were summarised as mean±standard deviation (SD), while skewed variables were expressed as median (interquartile range). Categorical variables were summarised as frequencies and percentages. Group comparisons (with vs. without DPN) were made using Student's t-test or Mann–Whitney U test for continuous variables and chi-square or

Fisher's exact test for categorical variables. Correlation analyses were performed using Pearson's or Spearman's correlation coefficients depending on distribution to explore relationships between TyG index, other metabolic markers. Diagnostic performance of the TyG index, ACR, HbA1c, uric acid and Apo A1 for predicting DPN was assessed using receiver operating characteristic (ROC) curve analysis. The area under the curve (AUC) with 95% confidence intervals was calculated.

In addition to the primary analyses, subgroup analyses were conducted to explore the relationship between the TyG index and other metabolic markers across clinically relevant strata of patients with T2DM. Specifically, comparisons were made by gender, age categories and duration of diabetes. These analyses were intended to identify whether the predictive or diagnostic utility of the TyG index was modified by demographic or clinical characteristics of the study population. All analyses were two-tailed and a p value <0.05 was considered statistically significant.

RESULTS

A total of 102 participants were recruited into the study, out of which 61.8% (n=63) had DPN. Table 1 shows the socio-demographic, clinical and biochemical characteristics of participants with and without DPN. Participants with DPN had significantly lower frequency of physical activity (47.6% versus 30.8%, p=0.012), lower diastolic blood pressure (mean±SD: 79.84±11.87 mmHg versus 84.77±7.05 mmHg, p=0.021) but higher prevalence of comorbid hypertension (81.0% versus 59.0%, p=0.022) and longer duration of diabetes (for instance, 31.7% of the 11–15 years category versus 7.7%; p=0.007) compared to those without DPN.

Furthermore, patients with DPN had significantly lower Apo A-I levels (median 109.6, IQR: 98.5–123.6 versus 118.4, IQR: 113.0–123.2; p=0.013), higher urine albumin (median 47.5, IQR: 21.6–101.9 versus 20.1, IQR: 11.5–32.8; p<0.001), higher serum creatinine (median 90.1, IQR: 70.0–104.8 versus 78.6, IQR: 63.5–94.6; p=0.048) and higher albumin-to-creatinine ratio (median 4.3, IQR: 1.5–7.3 versus 1.5, IQR: 0.8–2.1; p<0.001). Estimated GFR was also lower in participants with DPN (median 75.1, IQR: 66.4–91.7 versus 81.0, IQR: 73.2–96.2; p=0.017). Although the TyG index was slightly lower among participants with DPN (median 9.3, IQR: 8.7–9.8) compared to those without DPN (median 9.6, IQR: 9.1–9.9), this difference did not reach statistical significance (p=0.218). Other socio-demographic and biochemical variables, including age, sex, education, income, lipid profile, HbA1c, uric acid and lifestyle factors, showed no statistically significant differences between groups.

Table 2 presents the correlation between TyG index and some potential metabolic markers of DPN. The TyG index showed weak, non-significant correlations with HbA1c (ρ=0.162, p=0.103), Apo A-I (ρ=–0.184, p=0.064) and ACR (ρ=0.141, p=0.157) (Table 2). In contrast, uric acid correlated positively with HbA1c (ρ=0.377, p<0.001) and ACR (ρ=0.258, p=0.009), while inversely with Apo A-I (ρ=–0.327, p=0.001); all other associations were not statistically significant.

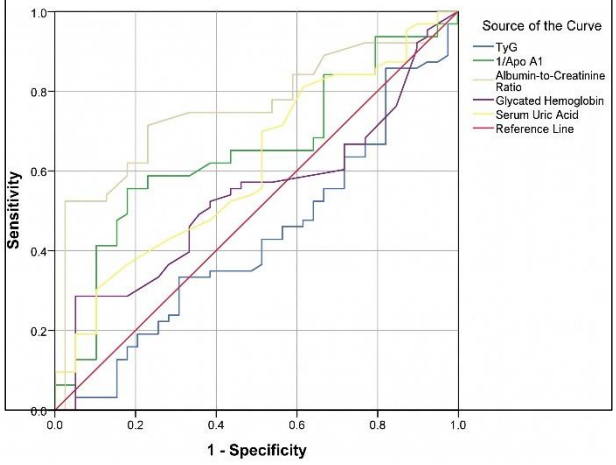

Figure 1: Receiver operating characteristic (ROC) curves for the TyG index, Apo A-I, ACR, HbA1c and Uric Acid in discriminating DPN among Nigerian patients with type 2 diabetes mellitus.

Table 3 and Figure 1 present the diagnostic performance of the TyG index and other metabolic markers for identifying diabetic peripheral neuropathy. The TyG index demonstrated poor diagnostic performance for DPN with an AUC of 0.427 (95% CI: 0.313–0.542, p=0.218), which was not statistically significant. In contrast, Apo A-I showed significantly poor–acceptable performance with an AUC of 0.647 (95% CI: 0.538–0.757, p=0.013), while ACR demonstrated a significant and acceptable diagnostic accuracy with an AUC of 0.757 (95% CI: 0.663–0.852, p<0.001). Other biomarkers, including HbA1c and uric acid, did not show statistically significant predictive value. The TyG index demonstrated poor performance in discriminating participants with DPN and was therefore not suitable for further assessment of diagnostic performance and cut-off determination.

Tables 4-6 present findings from the additional subgroup analyses. The TyG index did not differ significantly across age (median 9.37, IQR: 8.43–10.88 vs 9.45, IQR: 8.35–10.48 vs 9.30, IQR: 7.74–10.44; p=0.593) or gender (median 9.36, IQR: 8.82–9.77 vs 9.50, IQR: 8.67–9.96; p=0.559). By contrast, TyG varied significantly with diabetes duration, showing a progressive rise from median 9.1 (IQR: 8.6–9.6) in those with <5 years of diabetes to 9.6 (IQR: 9.2–10.5) in those with >16 years (p=0.001).

Other significant subgroup findings included lower Apo A-I in participants >60 years (median 109.4 mg/dl, IQR: 75.6–169.9) compared to those aged 51–60 years (median 119.7 mg/dl, IQR: 80.3–169.3; p=0.008); lower Apo A-I in males (median 106.2 mg/dL, IQR: 83.6–122.1) compared to females (median 117.4 mg/dl, IQR: 105.5–

124.8; p=0.005); and a further decline in Apo A-I with increasing diabetes duration, from median 116.0 mg/dl (IQR: 101.3–124.4) in<5 years to 106.0 mg/dl (IQR: 95.0–118.0) at >16 years (p=0.007). In addition, serum uric acid was higher in males (0.39±0.12 mmol/l) compared to females (0.31±0.10 mmol/l, p=0.001). All other subgroup comparisons, including HbA1c across age, sex and diabetes duration, showed no statistically significant differences.

Table 1: Socio-demographic, clinical and biochemical characteristics of participants with and without DPN.

Characteristics	Without DPN (N=39), Mean±SD, Median (IQR) or n (%)	With DPN (N=63), Mean±SD, Median (IQR) or n (%)	P value
Socio-demographic			
Age in years	56.10±8.38	59.95±10.43	0.054
Sex			
Female	27.0 (69.2)	39.0 (61.9)	0.525
Male	12.0 (30.8)	24.0 (38.1)	
Educational status			0.993
No formal education	12.0 (30.8)	21.0 (33.3)	
Primary	8.0 (20.5)	13.0 (20.6)	
Secondary	6.0 (15.4)	9.0 (14.3)	
Tertiary	13.0 (33.3)	20.0 (31.7)	
Marital status			0.058
Single	2.0 (5.1)	0.0 (0.0)	
Married	31.0 (79.5)	44.0 (69.8)	
Widowed	6.0 (15.4)	19.0 (30.2)	
Employment status			0.180
Unemployed	5.0 (12.8)	18.0 (28.6)	
Employed	27.0 (69.2)	36.0 (57.1)	
Retired	7.0 (17.9)	9.0 (14.3)	
Income estimate			0.153
Nil	5.0 (12.8)	15.0 (23.8)	
<₦50,000	17.0 (43.6)	33.0 (52.4)	
₦50,001–₦100,000	14.0 (35.9)	11.0 (17.5)	
₦100,001–₦200,000	3.0 (7.7)	4.0 (6.3)	
Tribe			0.996
Tiv/Etilo	28.0 (71.8)	46.0 (73.0)	
Idoma/Igede	6.0 (15.4)	10.0 (15.9)	
Others	5.0 (12.8)	7.0 (11.1)	
Religion			0.199
Christian	35.0 (89.7)	61.0 (96.8)	
Muslim	4.0 (10.3)	2.0 (3.2)	
Alcohol intake			0.062
Yes	19.0 (48.7)	43.0 (68.3)	
No	20.0 (51.3)	20.0 (31.7)	
Dietary modification			0.360
Not at all	20.0 (51.3)	35.0 (55.6)	
Seldom	10.0 (25.6)	13.0 (20.6)	
Occasionally	9.0 (23.1)	11.0 (17.5)	
Regularly	0.0 (0.0)	4.0 (6.3)	
Exercise/physical activity			0.012*
Not at all	12.0 (30.8)	30.0 (47.6)	
Seldom	16.0 (41.0)	8.0 (12.7)	
Occasionally	9.0 (23.1)	19.0 (30.2)	
Regularly	2.0 (5.1)	6.0 (9.5)	
Smoking			0.084
Yes	1.0 (2.6)	9.0 (14.3)	
No	38.0 (97.4)	54.0 (85.7)	
Clinical			
Systolic BP	130.0 (120–140)	130.0 (120–140)	0.204
Diastolic BP	84.77±7.05	79.84±11.87	0.021*
Comorbid hypertension			0.022*
Yes	23.0 (59.0)	51.0 (81.0)	
No	16.0 (41.0)	12.0 (19.0)	

Continued.

Characteristics	Without DPN (N=39), Mean±SD, Median (IQR) or n (%)	With DPN (N=63), Mean±SD, Median (IQR) or n (%)	P value
Duration of DM (in years)			0.007*
<5	21.0 (53.8)	15.0 (23.8)	
6–10	10.0 (25.6)	23.0 (36.5)	
11–15	3.0 (7.7)	20.0 (31.7)	
16–20	3.0 (7.7)	2.0 (3.2)	
≥20	2.0 (5.1)	3.0 (4.8)	
Biochemical			
Fasting plasma glucose (mmol/l)	7.1 (5.9–12.7)	7.6 (5.9–9.8)	0.836
Glycated haemoglobin (HbA1c) (%)	6.1 (5.2–7.4)	7.0 (5.4–8.3)	0.539
Total cholesterol (mmol/l)	4.2 (3.3–5.5)	3.8 (3.2–4.4)	0.194
Triglyceride (mmol/l)	2.31 (1.3–3.3)	2.00 (1.3–2.7)	0.096
HDL hcolesterol (mmol/l)	1.11±0.26	1.06±0.32	0.441
LDL cholesterol (mmol/l)	1.64 (0.8–3.2)	1.91 (0.9–2.6)	0.632
Triglyceride-glucose (TyG) index	9.6 (9.1–9.9)	9.3 (8.7–9.8)	0.218
Apoprotein A-I	118.4 (113.0–123.2)	109.6 (98.5–123.6)	0.013*
Urine albumin	20.1 (11.5–32.8)	47.5 (21.6–101.9)	<0.001*
Serum creatinine	78.6 (63.5–94.6)	90.1 (70.0–104.8)	0.048*
Urine creatinine	11.3 (7.2–21.0)	12.4 (6.4–18.3)	0.513
Serum uric acid	0.31±0.11	0.35±0.11	0.058
Albumin-to-creatinine ratio	1.5 (0.8–2.1)	4.3 (1.5–7.3)	<0.001*
eGFR (CKD-EPI) (ml/min/1.73m²)	81.0 (73.2–96.2)	75.1 (66.4–91.7)	0.017*

*Significant at p≤0.05.

Table 2: Correlation matrix of TyG index and some selected metabolic markers of T2DM complications.

Variables	TyG	HbA1c	Apo A-I	Uric acid	ACR
TyG	1.000				
HbA1c	ρ=0.162 (p=0.103)	1.000			
Apo A-I	ρ=–0.184 (p=0.064)	ρ=–0.020 (p=0.846)	1.000		
Uric acid	r=0.089 (p=0.373)	ρ=0.377** (p<0.001)	ρ=–0.327** (p=0.001)	1.000	
ACR	ρ=0.141 (p=0.157)	ρ=0.194 (p=0.051)	ρ=0.094 (p=0.345)	ρ=0.258** (p=0.009)	1.000

ρ=Spearman's rho (used when ≥1 variable skewed), r=Pearson's correlation (only when both normally distributed); * Significant at p<0.05, ** Significant at p<0.01.

Table 3: Diagnostic accuracy of selected metabolic markers for DPN in the study population.

Biomarker	AUC	SE	95% CI	P value	Interpretation
TyG index	0.427	0.058	0.313–0.542	0.218	Poor, not significant
Apo A-I	0.647	0.056	0.538–0.757	0.013*	Poor-acceptable, significant (inverse performance).
ACR	0.757	0.048	0.663–0.852	<0.001*	Acceptable, statistically significant
HbA1c	0.536	0.058	0.423–0.649	0.540	Poor, not significant
Uric acid	0.612	0.057	0.501–0.723	0.058	Poor–acceptable, borderline significance

*Significant at p≤0.05.

Table 4: Comparison of TyG index and other metabolic markers across age categories.

Variable	<50 years (n=20) Mean±SD, Median (IQR)	51-60 years (n=32) Mean±SD, Median (IQR)	>60 years (n=50) Mean±SD, Median (IQR)	P value
TyG index	9.37 (8.43–10.88)	9.45 (8.35–10.48)	9.30 (7.74–10.44)	0.593
HbA1c (%)	6.90 (4.0–10.0)	7.04 (4.0–13.2)	6.30 (4.0–9.0)	0.300
Apo A-I (mg/dl)	116.36 (77.9–149.7)	119.68 (80.3–169.3)	109.43 (75.6–169.9)	0.008*
ACR (mg/g)	2.06 (0.50–6.40)	9.28 (0.20–58.0)	6.61 (0.20–73.1)	0.126
Uric Acid (mmol/l)	0.33±0.12	0.32±0.11	0.35±0.11	0.546

*Significant at p≤0.05.

Table 5: Comparison of TyG index and other metabolic markers across gender categories.

Variable	Female (N=66) Mean±SD or Median (IQR)	Male (N=36) Mean±SD or Median (IQR)	P value
TyG index	9.36 (8.82–9.77)	9.50 (8.67–9.96)	0.559
HbA1c (%)	6.95 (5.35–7.60)	6.00 (5.00–7.88)	0.445
Apo A-I (mg/dl)	117.4 (105.5–124.8)	106.2 (83.6–122.1)	0.005*
ACR (mg/g)	2.20 (0.88–4.40)	2.05 (0.70–7.20)	0.844
Uric Acid (mmol/l)	0.31±0.10	0.39±0.12	0.001*

*Significant at p≤0.05.

Table 6: Comparison of TyG index and other metabolic markers across duration of diabetes categories.

Variable	<5 years (n=21) Median (IQR) or Mean±SD	6–10 years (n=41) Median (IQR) or Mean±SD	11–15 years (n=21) Median (IQR) or Mean±SD	>16 years (n=19) Median (IQR) or Mean±SD	P value
TyG index	9.1 (8.6–9.6)	9.5 (9.0–9.8)	9.4 (9.0–9.9)	9.6 (9.2–10.5)	0.001*
HbA1c (%)	6.6 (6.2–8.7)	7.3 (6.4–9.2)	7.5 (6.2–9.8)	7.2 (6.2–9.8)	0.269
Apo A-I (mg/dl)	116.0 (101.3–124.4)	114.0 (101.0–122.0)	112.0 (98.0–121.0)	106.0 (95.0–118.0)	0.007*
ACR (mg/g)	2.2 (0.8–4.9)	2.4 (0.9–7.1)	3.0 (1.2–8.0)	7.8 (3.0–30.0)	0.107
Uric Acid (mmol/l)	0.34±0.09	0.33±0.09	0.34±0.08	0.36±0.08	0.434

*Significant at p≤0.05.

DISCUSSION

This study demonstrated a high prevalence of diabetic peripheral neuropathy among patients attending a tertiary diabetes clinic. Individuals with DPN tended to have longer diabetes duration, a higher frequency of hypertension, lower physical activity levels and worse renal parameters. Although the TyG index did not differ significantly between participants with and without neuropathy and showed limited diagnostic performance for DPN, it increased with longer diabetes duration, suggesting that it may reflect cumulative metabolic stress rather than neuropathy risk per se. In contrast, Apo A-I concentrations were lower in patients with DPN and displayed modest discriminatory ability, while ACR showed acceptable performance as a marker of neuropathy.

These findings are consistent with current concepts of DPN pathogenesis. Diabetic neuropathy arises from a complex interplay of metabolic and microvascular injury, in which chronic hyperglycaemia, oxidative stress, inflammation, advanced glycation end-products and endothelial dysfunction compromise the vasa nervorum and neural tissue, resulting in both small- and large-fibre damage. Albuminuria, as captured by ACR, is a sensitive indicator of systemic endothelial and microvascular injury and has been linked in previous studies to peripheral nerve dysfunction.[25,26] Low Apo A-I may further exacerbate this vulnerability by diminishing antioxidant, anti-inflammatory and endothelial-protective effects, thereby promoting progression of neuropathy.[27] Elevated serum uric acid, which correlated with HbA1c and ACR in this cohort, can intensify oxidative and inflammatory stress, contributing to microvascular injury and nerve damage.[28,29] Although the TyG index is a validated marker of insulin resistance and cardiometabolic risk, its poor discriminatory value for DPN in this study suggests that neuropathy reflects broader and more heterogeneous mechanisms than insulin resistance alone.[30,31]

The prevalence of DPN and the observed associations in this cohort are in keeping with published evidence. Pooled analyses and systematic reviews have documented a substantial burden of DPN across Africa, with reported prevalence around 46% and wide variation according to diagnostic criteria, while studies from Nigeria similarly show broad ranges that reflect both methodological differences and a genuinely high disease burden.[32,33] The prevalence observed here lies within the upper end of Nigerian estimates and is consistent with the referral-based nature of tertiary hospital populations. In line with multiple prior reports, albuminuria and ACR were strongly associated with neuropathy, supporting their role as markers of generalized microvascular disease as well as renal injury.[25,26]

The behaviour of the other biochemical indices in this study mirrors the heterogeneity of the existing literature. Although the TyG index has been repeatedly linked to insulin resistance and macrovascular outcomes, evidence for a consistent association with microvascular complications such as DPN remains limited, which accords with the lack of significant discriminatory value observed here.[30,31] The finding that TyG values increased with longer diabetes duration supports its role as an indicator of chronic metabolic burden and cumulative cardiometabolic risk.[34] By contrast, Apo A-I and HDL-related pathways are increasingly recognised as important modulators of diabetic complications, with lower Apo A-I

or dysfunctional HDL associated with adverse outcomes in diabetic foot disease and impaired tissue repair; the reduced Apo A-I levels observed in patients with DPN in this study align with these reports.[27,35] Similarly, accumulating evidence links higher serum uric acid to microvascular complications, including neuropathy, which is consistent with the observed correlations between uric acid, glycaemic burden and albuminuria.[36-38]

These findings have important implications for diabetes care in resource-limited Nigerian settings. Given the high prevalence of neuropathy in hospital-based cohorts and its association with longer diabetes duration, hypertension and albuminuria, systematic screening using simple validated tools such as the MNSI, 10g monofilament and tuning fork remains essential.[17,33] Markers such as ACR, which reflect widespread microvascular injury, may help identify patients who would benefit from closer surveillance, structured foot-care education, podiatry referral and intensified cardiovascular risk management.[25,39] In contrast, although the TyG index remains useful for cardiometabolic risk stratification, its limited value for identifying DPN in this cohort reinforces the view that neuropathy is driven by mechanisms that extend beyond insulin resistance alone.[26] The associations observed with modifiable factors such as blood pressure control, renal protection and physical activity further emphasise the importance of comprehensive lifestyle and risk-factor management in reducing neuropathy burden and preventing severe outcomes such as ulceration and amputation.[32]

The study also has limitations that should be considered when interpreting the results. Strengths include the use of validated bedside neuropathy assessments, which are particularly appropriate for low-resource settings, standardized biochemical testing with external quality assurance and the comparative evaluation of multiple metabolic markers. However, the cross-sectional design limits causal inference and prevents assessment of temporal biomarker changes, while the modest sample size restricts power for subgroup analyses. Recruitment from tertiary clinics may have enriched the sample for more advanced disease, reducing generalisability to community populations. In addition, the absence of nerve conduction studies may have resulted in misclassification of borderline cases. Future prospective studies in larger Nigerian cohorts are needed to validate ACR and Apo A-I as predictors of incident DPN, establish clinically useful thresholds, clarify mechanistic links among insulin resistance, HDL dysfunction, uric acid and microvascular injury and evaluate practical strategies for early detection and prevention in routine care.

CONCLUSION

This study examined the association between the triglyceride–glucose index and diabetic peripheral neuropathy among Nigerian adults with type 2 diabetes attending a tertiary care facility. Although the burden of neuropathy was substantial, the TyG index demonstrated limited ability to distinguish patients with and without neuropathy. In contrast, longer duration of diabetes, coexisting hypertension, reduced physical activity, albuminuria and lower apolipoprotein A-I levels were significantly associated with neuropathic involvement. These findings underscore the importance of routine neuropathy screening and the implementation of focused preventive strategies in clinical practice. While the use of validated neuropathy assessment tools and standardized biochemical assays strengthens the study, the cross-sectional design, hospital-based recruitment and modest sample size constrain causal interpretation and generalizability. Larger prospective studies, particularly in community settings, are therefore required to confirm the predictive value of candidate biomarkers and to guide effective approaches for early detection and prevention of diabetic peripheral neuropathy.

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